European Heart Journal (2008) **29**, 71–78
doi:10.1093/eurheartj/ehm555

Sinus node disease and arrhythmias in the long-term follow-up of former professional cyclists

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Received 6 February 2007; revised 16 October 2007; accepted 5 November 2007; Online publish-ahead-of-print 7 December 2007

Aims Significant brady- and tachyarrhythmias may occur in active endurance athletes. It is controversial whether these arrhythmias do persist after cessation of competitive endurance training.

Methods and results Among all 134 former Swiss professional cyclists [hereafter, former athletes (FAs)] participating at least once in the professional bicycle race Tour de Suisse in 1955–1975, 62 (46%) were recruited for the study. The control group consisted of 62 male golfers matched for age, weight, hypertension, and cardiac medication. All participants were screened with history, clinical and echocardiographic examination, ECG, and 24 h ECG. The time for the last bicycle race of FAs was 38 ± 6 years. The mean age at examination was 66 ± 6 years in controls and 66 ± 7 years in FAs ($P = 0.47$). The percentage of study participants with >4 h current cardiovascular training per week was identical. QRS duration (102 ± 20 vs. 95 ± 13 ms, $P = 0.03$) and corrected QTc interval (416 ± 27 vs. 404 ± 18, $P = 0.004$) were longer in FAs. There was no significant difference in the number of isolated atrial or ventricular premature complexes, or supraventricular tachycardias in the 24 h ECG; however, ventricular tachycardias tended to occur more often in FAs than in controls (15 vs. 3%, $P = 0.05$). The average heart rate was lower in FAs (66 ± 9 vs. 70 ± 8 b.p.m.) ($P = 0.004$). Paroxysmal or persistent atrial fibrillation or flutter was reported more often in FAs ($P = 0.028$). Sinus node disease (SND), defined as bradycardia of <40 b.p.m. (10 vs. 2%), atrial flutter (6 vs. 0%), pacemaker for bradyarrhythmias (3 vs. 0%), and/or maximal RR interval of >2.5 s (6 vs. 0%), was more common in FA (16%) than in controls (2%, $P = 0.006$). Observed survival of all FAs was not different from the expected.

Conclusions Among FAs, SND occurred significantly more often compared with age-matched controls, and there is trend towards more frequent ventricular tachycardias. Further studies have to evaluate prevention of arrhythmias with extreme endurance training, the necessity of regular follow-up of heart rhythm, and management of arrhythmias in former competitive endurance athletes.

Keywords Cyclists • Sinus node disease • Arrhythmias • Endurance training • Atrial fibrillation • Atrial flutter

Introduction

The effect of chronic high-intensity endurance exercise may result in cardiac changes called 'athlete's heart', including typical morphological changes and a slow heart rate with significant brady- and tachyarrhythmias.[1–7] In active athletes, first degree and second degree atrioventricular (AV) block type, Wenckebach, are not of concern; however, Mobitz type II second degree AV block or complete heart block are rarely seen.[8,9]

It has been debated whether changes in cardiac structure and function persist in elderly endurance athletes, and whether there is a higher prevalence of arrhythmic complications or the need

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for pacemaker therapy. Some studies found an increased frequency of supraventricular and ventricular premature complexes during and after exercise in middle aged to elderly athletes.[10,11] Also, a higher prevalence of ventricular arrhythmias and sinus bradycardia in elderly male athletes with a lifelong history of regular physical exercise[12] or 'lone' atrial fibrillation in vigorously exercising middle-aged men have been reported.[9,13] A 12 year follow-up study of 20 veteran athletes reported on two former endurance athletes with atrial fibrillation combined with complete heart block and 15 s asystole necessitating pacemaker implantation.[14] Profound bradyarrhythmias in elderly athletes because of sinus node disease (SND) may increase the risk of sudden cardiac death.[9] Implantation of a permanent pacemaker has been reported in up to 11% of former marathon runners in a small study.[14] So far, however, there is no large study on the problem of arrhythmias or SND nor is there any case-matched control study on arrhythmias in athletes in the long-term follow-up.

The aim of our study was to evaluate ECG changes, arrhythmias, and signs of SND in the long-term follow-up 30–50 years after year-long high endurance training in former professional cyclists (FAs) by comparing them to case-matched controls who had never performed any high-endurance competition.

Methods

Study subjects

The study groups consisted of 62 male FAs, aged 66 ± 6 years and 62 male controls, aged 66 ± 7 years, who never performed professional endurance training. All cyclists had to have at least participated once in the professional cycling race Tour-de-Suisse during the years 1955 until 1975. Among these 134 cyclists, 24 have died (cause of death unknown in seven, cancer in four, car accident in three, leukaemia in two, suicide in two, chronic hepatitis/liver failure in two, coronary artery disease in two, cerebral haemorrhage in one, and alcohol abuse in one). The median age at death was 62 ± 12 years (median, 67 years; range, 39–79 years) in 21 of 24 deceased FAs. Seven persons were living abroad, 12 could not be traced, and 29 did not want to participate in the study. Thus, 62 (46%) agreed to participate in the study. The controls were selected among 309 male senior leisure time golfers consisting of all senior golfers from the database of two regional golf clubs, who never performed in high-endurance training. All golfers were contacted with a letter where they had to fill in data regarding whether they wanted to participate, their age, height, weight, and other details including information on current and previous physical exercise. Among the 165 men who agreed to participate, 62 were frequency matched for age, body mass index, hypertension, and the current hours of physical training. Smoking was more common in controls and could not be matched. Previous smoking was significantly more common in golfers ($P < 0.0001$), there was no significant difference in the occurrence of hyperlipidemia ($P = 0.32$), diabetes ($P = 1.0$), or positive family history for coronary artery disease ($P = 0.24$).

The cyclists were competitors for 11 ± 4 years and stopped their professional time with participating in competition 38 ± 7 years ago (median, 38 years; range, 15–49 years). During their professional time, they cycled an estimate of 25 200 ± 9700 km/year (median, 25 000; range, 9000–40 000) maximally. One bicycle year was defined as riding 1000 km on the bicycle per year. FAs reported an estimate of 225 ± 140 (median, 196 years; range, 60–730 years) bike years in their professional time and 119 ± 116 bike years (median, 100 years; range, 0–625 years) after they stopped competition. Overall, the FAs had achieved 342 ± 181 bike years (median, 311 years; range, 60–975 years), compared with 12 ± 21 bike years (median, 3 years; range, 0–120 years) in the control group ($P < 0.0001$).

All hours of sport-related energy expenditure were counted per week including swimming, jogging, playing tennis, rowing, or riding the bicycle. We divided the hours of walking or playing golf (without carts) by four to make energy expenditure and cardiovascular effect comparable with cycling or running. The current hours were 4.6 ± 4.4 (median, 4 h; range, 0–20 h) in FAs vs. 3.3 ± 1.7 h in controls (median, 3 h; range, 1–8 h) ($P = 0.03$). There was no significant difference in persons with more than 4 h of sport-related cardiovascular training per week: 52% of FAs vs. 44% of controls ($P = 0.47$). However, at the time of this study, the FAs were cycling more than controls with 3882 ± 4170 km per year (median, 2500 years; range, 0–17 000 years) compared with 348 ± 862 km per year (median, 0; range, 0–4500) in controls ($P < 0.0001$). Contrarily, controls were significantly more often jogging and rowing. In addition to the electrocardiographic and echocardiographic examinations, a detailed patient history using a questionnaire and an interview were obtained.

The study was approved by the local ethics committee and informed consent was obtained from all study participants.

A total of 44 (71%) FAs admitted the use of any performance enhancing agents including amphetamines in 20 of 44 (45%), stenamine (another amphetamine) in seven (16%), coramine in five (11%), and anabolic steroids in four (9%). Other performance enhancing agents used occasionally included wine and ephedrine.

Electrocardiogram at rest and Holter electrocardiogram

A 12-lead resting electrocardiogram (EGC) was recorded for each participant. For the ambulatory Holter ECG, digital three-channel ECG recorder was used (Lifecard CF from DelmarReynolds, UK). The exploring electrodes were placed on the middle of the sternum and on the fifth rib at the left and the right anterior axillary line. The recording quality was checked for stability and the capability to detect P-waves. Participants were instructed to undertake a usual strenuous physical activity and to keep a diary for the registration period. Holter recordings were analysed with commercial software (Pathfinder V 8.602 from DelmarReynolds, UK). All findings of the ECG and Holter ECG were reviewed by U.B.

Definitions

Supraventricular tachycardias and ventricular tachycardias were defined as a sudden increase of three or more following beats at a rate of >120 b.p.m.

The frequency of atrial premature complexes (APCs) or ventricular premature complexes (VPCs) was defined as: none, $<1\ h^{-1}$; rare, ≥1 and <248/24 h; occasional, ≥248 and <1439/24 h; frequent, ≥1440/24 h.

Definition of sinus node disease

The presence of SND was determined in 116 subjects without β-blockers (eight with β-blocker therapy excluded). SND was defined by the presence of more than one of the following criteria: an average heart rate <50 b.p.m. at day-time or <40 b.p.m. at night and/or maximal RR interval of at least 2.5 s, atrial flutter, or pacemaker implantation for SND.[15]

Echocardiography

A complete two-dimensional and Doppler echocardiographic examination was performed in all subjects according to the recommendations of the American Society of Echocardiography,[16] including two-dimensionally guided M-mode measurements using 512 Acuson Sequoia machines and Aplio 80 Toshiba. Echocardiography was performed on the same day as the resting ECG and the commencement of the Holter ECG recording. Left ventricular muscle mass index was calculated using the Devereux-modified American Society of Echocardiography cube equation.[17] Data of left ventricular ejection fraction and left ventricular muscle mass index were used for this study. All studies were digitally stored and sent to C.A.J. for subsequent review, if necessary.

Statistics

Descriptive statistics include frequencies and percentages for categorical data, mean ± SD for approximately normally distributed data and additionally median [range] for markedly non-normally distributed data. Fisher's exact test and χ^2 test were used to compare the categorical data between FAs and controls. The Mann–Whitney test was used to compare the continuous data. Correlations between continuous variables were analysed using Spearman's rank correlation. A *P*-value of <0.05 was considered to be statistically significant. All reported *P*-values are two-sided and have not been adjusted for multiple testing. Data were analysed using SPSS 11 (SPSS inc., Chicago, IL, USA). Survival of 119 former participants of the Tour de Suisse was compared with the expected survival curve of a male age- and calendar time-matched Swiss reference population.

Results

Patient characteristics and symptoms

Clinical details of both groups are shown in *Table 1*. There was no significant difference between the two groups in age, body mass index, and cardiovascular risk factors—apart from a history of smoking. Dizziness was the only cardiac symptom more common in FAs ($P = 0.02$).

There was no significant difference between the two groups regarding the occurrence of syncope. A history of previous pacemaker implantation was present in two FAs for symptomatic SND with bradycardic atrial flutter.

Functional class was similar and most commonly limited because of obesity, hypertensive heart disease, and obstructive lung disease as a result of smoking.

The intake of cardiac medication did not differ significantly between both groups. A total of 45 FAs (73%) and 47 (76%) controls did not take any of these drugs.

Electrocardiographic data

Resting ECG findings are shown in *Table 2*. The resting ECG showed a slightly lower heart rate in FA than in controls, as well as a longer QRS duration and corrected QT interval. There was no significant difference in right or left bundle branch block, left anterior or posterior hemiblock or atrioventricular block. However, chronic or paroxysmal atrial fibrillation or flutter occurred significantly more common in FA ($P = 0.0028$).

In the Holter ECG (see *Table 3*), FAs had a significantly lower average heart rate ($P = 0.004$). The minimal heart rate in the 24 h ECG tended to be lower in FAs ($P = 0.05$). Bradycardia with heart rates <50 b.p.m. during the day occurred significantly more often in FAs than in controls ($P = 0.004$). Two FAs had also sinus bradycardia <40 b.p.m. during the day. Bradycardic episodes were asymptomatic in all FAs.

Four athletes (6%) vs. 0% of the controls had a maximal RR interval exceeding 2.5 s ($P = 0.12$). The maximal RR interval was significantly longer in the cyclists than in controls ($P = 0.008$). One FA had an asymptomatic sinus pause of 5.3 s; overall, he had 192 pauses >2.5 s.

Sinus node disease as defined above was more common in FAs than in controls ($P = 0.004$). There was no significant difference in the group of FAs with or without SND regarding age, number of bicycle years, or current hours of endurance training. However, the length of previous competitive years was slightly longer, 13 ± 6 years in FAs with signs of SND vs. 10 ± 3 years in those without ($P = 0.06$).

Table 1 Clinical findings of the 62 former professional cyclists and 62 controls

	FAs ($n = 62$)	Controls ($n = 62$)	*P*-value
Age, years	66 ± 7	66 ± 6	0.47
Body mass index, kg/m^2	25.6 ± 2.8	25.9 ± 2.4	0.58
Hypertension, *n*(%)	22 (35)	18 (29)	0.56
Current medication			
Beta blocker, *n*(%)	5 (8)	3 (5)	0.72
ACEI/ARB, *n*(%)	11 (18)	10 (16)	1.0
Calcium-channel blocker,*n*(%)	3 (5)	2 (3)	1.0
Diuretic, *n*(%)	7 (11)	4 (6)	0.53
Angina pectoris	3 (5)	1 (2)	0.62
Dyspnea on exertion			
NYHA class I, *n*(%)	53 (86)	55 (89)	0.80
NYHA class II, *n*(%)	7 (11)	6 (10)	
NYHA class III/IV, *n*(%)	2 (3)	1 (2)	
Syncope, ever, *n*(%)	7 (11)	2 (3)	0.44
Dizziness, *n*(%)	17 (27)	6 (10)	0.02
Palpitations, *n*(%)	8 (13)	8 (13)	1.0
Pacemaker, *n*(%)	2 (3)	0	0.50
Heart failure ever, *n*(%)	2 (3)	0	0.50
Myocardial infarction, *n*(%)	3 (5)	1 (2)	0.62
Atrial flutter or fibrillation, *n*(%)	6 (10)	0	0.028
Atrial flutter, persistent	3		
Atrial flutter, paroxysmal	1		
Atrial fibrillation, persistent	2		
Atrial fibrillation, paroxysmal	0		

FAs, former athletes; BSA, body surface area; NYHA, New York Heart Association; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker.

Table 2 Comparison of findings of resting ECG

	FAs ($n = 62$)	Controls ($n = 62$)	*P*-value
Heart rate, b.p.m.	58 ± 10	63 ± 9	0.01
PR interval, ms	186 ± 37	177 ± 24	0.14
QRS duration, ms	102 ± 20	95 ± 13	0.027
Corrected QT interval, ms	416 ± 27	404 ± 18	0.004
Left bundle branch block	0	0	
Left anterior hemiblock, *n* (%)	3 (5)	3 (5)	1.0
Left posterior hemiblock, *n* (%)	0	1 (2)	1.0
RBBB complete, *n* (%)	0	3 (5)	0.24
RBBB incomplete, *n* (%)	2 (3)	1 (2)	1.0
AV block, *n* (%)	8 (13)	9 (15)	1.0
First degree	7 (11)	8 (13)	
Second degree	0	1 (2)	
Third degree, *n* (%)	1 (2)	0	
Atrial flutter or fibrillation, *n* (%)	6 (10)	0	0.028

RBBB, right bundle branch block; AV, atrioventricular; FAs, former athletes.

Table 3 Holter ECG findings in the two groups

	FAs ($n = 62$)	Controls ($n = 62$)	*P*-value
Heart rate, mean, b.p.m.	66 ± 9	70 ± 8	0.004
Heart rate, minimal, b.p.m.	49 ± 8	51 ± 6	0.05
Heart rate, maximal, b.p.m.	124 ± 26	124 ± 17	0.97
Heart rate <50 b.p.m. during the day, *n* (%)	20 (32)	6 (10)	0.004
Heart rate <40 b.p.m.			
Ever, *n* (%)	6 (10)	1 (2)	0.11
During the day, *n* (%)	2 (3)	0	0.49
During the night, *n* (%)	6 (10)	1 (2)	0.11
Maximal RR interval, ms (mean)	1761 ± 702	1499 ± 223	0.007
median (range)	1565 (1031–5300)	1500 (1100–2160)	
Maximal RR interval >2.5 s	4 (6%)	0	0.12

FAs, former athletes.

In FAs with atrial fibrillation or flutter, the total number of bicycle years was 504 ± 283 (median, 485; range, 243–975) vs. 327 ± 166 (median 290, range 60–720; $P = 0.04$) in those without.

Frequent and/or complex supraventricular or ventricular arrhythmias

There was no significant difference in the occurrence of atrial premature complexes or supraventricular tachycardias between the groups (see *Table 4*).

Table 4 Supraventricular arrhythmias

	FAs ($n = 62$)	Controls ($n = 62$)	*P*-value
Atrial premature complexes per 24 h			
Median (range)	18 (0–2616)	17 (1–6135)	0.35
Atrial premature complexes			
None <1/h, *n* (%)	35 (56)	39 (63)	
Rare, *n* (%)	23 (37)	19 (31)	
Occasional, *n* (%)	3 (5)	2 (3)	
Frequent, *n* (%)	1 (2)	2 (3)	
Number of subjects with SVT, *n* (%)	21 (34)	19 (31)	0.82
Number of runs of SVT			
Mean	0.7 ± 1.3	0.7 ± 1.4	0.88
Median (range)	0 (0–7)	0 (0–7)	

FAs, former athletes; SVT, supraventricular tachycardia.

Table 5 Comparison of ventricular arrhythmias

	FAs ($n = 62$)	Controls ($n = 62$)	*P*-value
Number of subjects with VPCs, *n* (%)	28 (45)	22 (35)	0.12
Number of VPCs per 24 h			
Total	337 ± 1054	390 ± 1211	0.59[a]
Median (range)	32 (0–7780)	67 (0–7792)	
None/rare VPCs	49	48	
Occasional VPCs	10	10	
Frequent VPCs	3	4	
Couplets of VPC			
Mean	12 ± 72	8 ± 33	0.70*
Median (range)	0 (0–569)	0 (0–222)	
Subjects with VT	9 (15%)	2 (3%)	0.05

FAs, former athletes VPCs; ventricular premature complexes; VT, ventricular tachycardia.
[a]Mann–Whitney test.

There was no significant difference in the percentage of subjects with VPCs or the number of VPCs between the groups (see *Table 5*). However, polymorphic ventricular premature complexes and VPC as bigemini and trigemini were more common in controls. Ventricular tachycardias tended to occur more often in FAs than in controls ($P = 0.05$). The length of VT ranged from three to 14 in FAs and three to eight in controls. The number of runs of VT ranged from one to 326 in FAs and from one to eight in controls.

Echocardiographic data

In FAs, the echocardiographic examination showed that the left ventricular ejection fraction was slightly lower than in controls (62 ± 8 vs. 64 ± 6%, $P = 0.049$). There was no significant difference in the thickness of the interventricular septum (11.1 ± 2.0

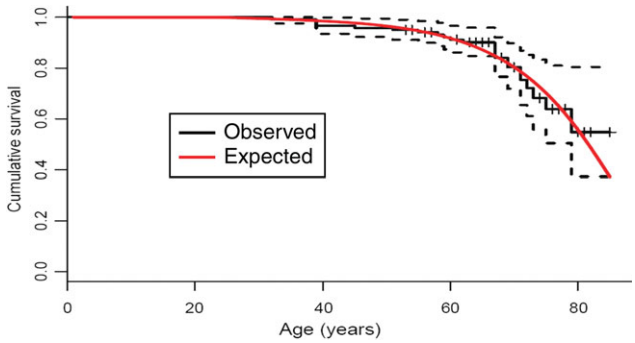

Figure 1 Survival curve of former athletes participating in the Tour de Suisse compared with a male age- and calendar time-matched Swiss reference population. The dotted lines represent a 95% pointwise confidence interval for the Kaplan–Meier estimate

in FAs vs. 11.2 ± 2.0 in controls, $P = 0.79$). The left ventricular muscle mass index tended to be larger in FAs with 110 ± 32 g/m^2 body surface area (median. 108; range, 51–213) vs. 101 ± 17 g/m^2 (median, 96; range, 61–142) in controls ($P = 0.07$). The LVEDD was 51 ± 4 mm in FAs vs. 49 ± 5 mm in controls ($P = 0.04$). Left and right atrial size indexed for body surface area was larger in FAs than in controls: left atrial volume index was 30 ± 13 ml/m^2 body surface area in 57 FAs in sinus rhythm (median, 27; range, 12–73) vs. 24 ± 8 ml (median, 32; range, 8–68) in 62 controls ($P = 0.02$); right atrial volume index was 29 ± 12 in 57 FAs in sinus rhythm (median, 26; range, 10–60) vs. 23 ± 8 in 62 controls (median, 22; range, 10–44) ($P = 0.002$).

Survival curve

Cumulative survival of 119 FA participating in the Tour de Suisse from 1955 to 1975 is shown in *Figure 1* as a Kaplan–Meier survival curve. Survival of FAs was similar to a matched Swiss male population.

Discussion

Our study suggests that extreme high endurance training might not only have physiological consequences such as signs of SND with bradycardia of <40 b.p.m., atrial flutter, prior pacemaker insertion for bradyarrhythmias and/or maximal RR interval of >2.5 s were more common in FAs (18%) than in controls (2%, $P = 0.004$). Also, in the FAs, the incidence of atrial flutter or fibrillation was higher and QRS duration and corrected QTc interval were longer at long-term follow-up.

Atrioventricular block and sinus node disease

Sinus bradycardia and atrioventricular conduction abnormalities including first and second-degree (Wenckebach) AV block represent part of the spectrum of arrhythmias in active endurance athletes.[6] Some of the changes are induced by increased parasympathetic and reduced sympathetic activity.[18] And also non-autonomic factors contribute to change in AV conduction. It was shown that sinus cycle length and sinus node recovery time were longer in endurance athletes after atropine and after propranolol, also the Wenckebach cycle and anterograde refractory period of the AV node.[18] In our FAs, there was no significant difference in PR intervals and no increase in the occurrence of AV block. This underlines that changes in AV conduction are mostly related to higher parasympathetic activity owing to their association with active training.[19] However, signs for intrinsic SND are present in the FA. Whereas in the active athlete, sinus bradycardia without symptoms is common and does not usually warrant further testing or treatment,[9] this is different in the elderly former high-endurance athlete. Clearly, SND seems to be a significant side-effect of high-endurance training performed during many years. Especially, its association with atrial flutter and atrial fibrillation and the need for pacemaker implantation suggest that it might also affect morbidity in these people. Our study is the largest on this topic with case-matched controls. The natural history study in SND depends on the underlying disease. However, there are a few data in the literature that raise concern on its impact on survival.[20] In a study with 35 untreated patients with SND aged ≥45 years, during a follow-up of 17 ± 15 months, 57% of patients had a cardiovascular event including syncope, heart failure, and tachyarrythmias.[20]

Atrial flutter or atrial fibrillation

Our former cyclists had significantly more atrial fibrillation/atrial flutter ($P = 0.028$). It has been described that vigorous long-term exercise is associated with atrial fibrillation in healthy middle aged men.[13,21] However, only few data are available. In one of these studies, the first attack of 'lone' atrial fibrillation in top veteran orienteers was at a mean age of 52 years (range 34–68 years) after an average training history of 36 years; there were also three episodes of atrial flutter.[13] Enhanced vagal tone and/or changes of the athlete's heart including enlarged atria and left ventricular hypertrophy may predispose the hearts to these arrhythmias.[13] Among patients with the so-called lone atrial fibrillation, the percentage of sportsmen is higher with 31–63 vs. 14–15% in the general population in several studies.[22,23] Most of these arrhythmias seem to be vagal (in 57%) occurring more commonly during sleep or after meals compared with 18% in

the non-sport patients.[22] Vagally induced atrial fibrillation was seen in 33–37% of male endurance athletes during a 9 year follow-up of atrial fibrillation.[24] In that study, older athletes presented with vagally induced atrial fibrillation more often than younger athletes. In our study, FA with a very high number of previous bicycle years had a higher left ventricular muscle mass, larger atria, and a significant higher occurrence of atrial fibrillation or flutter correlating with previous bicycle years indicating that there might be a threshold above which irreversible cardiac changes occur as another cause for atrial fibrillation or flutter. An association of a lifetime practice of >1500 h of sport was also associated with lone atrial fibrillation in another study, so there seems to be a threshold.[23]

In a study among 30 well-trained athletes with paroxysmal atrial fibrillation, permanent atrial fibrillation emerged in 17% of athletes, 10% of the 30 athletes had also atrial flutter.[24] In our athletes, atrial flutter was more common (in four FAs) than atrial fibrillation (in two FAs). In Hoogsteen's study, it has been suggested that persistent atrial fibrillation developed in only a minority of male endurance athletes.[24]

Significance of QRS and QTc prolongation

In the FAs, QRS duration and corrected QTc were slightly longer. This might be because of residual fibrosis of the myocardium, which is also reflected in the slightly higher left ventricular muscle that can be an explanation for the tendency of ventricular tachycardias to occur more often in FAs than in controls. So far, there are no autopsy data on the hearts of previous high-endurance athletes, which quantify fibrosis and other residual damage in these subjects. It has been shown in individuals with left ventricular hypertrophy secondary to essential hypertension that both QTc and QRS duration can correlate with left ventricular muscle mass[25,26] and may be predictive of ventricular arrhythmias and cardiovascular mortality as well.[25,27]

Ventricular arrhythmias

Frequent and/or complex ventricular arrhythmias occur frequently in trained athletes with physiological left ventricular hypertrophy.[11,28] These arrhythmias are very sensitive to deconditioning in athletes with and without structural heart disease: an impressive 80% decrease (from 10 611 ± 10 078 to 2165 ± 4877) has been described as well as a 90% decrease in the occurrence of non-sustained ventricular tachycardias with deconditioning.[28] However, ventricular arrhythmias in active endurance athletes do not necessarily represent a benign finding and can be because of changes in right ventricular structure or right ventricular arrhythmic involvement.[29] In our study, FAs tended to have only slightly more ventricular tachycardias than the controls. From this small difference, we cannot draw any definite conclusions and still assume that most ventricular arrhythmias are indeed reversible in athletes. It should be considered that the control group had significantly more smokers than the FA group, and it was shown by the Trial-II investigators that especially current smoking increases the incidence of fast ventricular tachycardia or ventricular fibrillation and may also be associated with supraventricular tachycardias.[30] Therefore, comparing FAs with a control group with fewer smokers might increase the relevance of a higher incidence of ventricular tachycardias in FA even more.

We do not have data on the impact of ventricular arrhythmias on survival in this group of FAs. The reported incidence of sudden death in athletes is low (1:200 000 to 1:300 000).[31]

Sudden death in the athletes is not usually because of acquired arrhythmias as a result of endurance training, but because of underlying congenital heart disease, coronary artery anomalies, and other rare causes such as cardiomyopathies or myocarditis, etc.[31,32] Still, the more frequent occurrence of ventricular tachycardias in FAs reinforces the need to investigate always the cause of death in cyclists. Besides, cardiac screening during and after high-level athletic participation should be performed in all former endurance athletes including cyclists, orienteers, rowers, marathon runners, triathletes, etc. with a resting ECG and—if this is abnormal and/or if there are cardiac symptoms such as palpitations, dizziness or syncope—by further examination by echocardiography, and Holter ECG.

Limitations

Cause of death in six of 24 deceased athletes is not known to us. Besides, 11 athletes could not be traced. This might have resulted in positive selection bias with underestimation of cardiac morbidity because of rhythm disturbances with stroke or sudden cardiac death. However, sudden cardiac death was not reported in 18 of 24 dead former cyclists in whom cause of death is known. Another limitation is that 32% (29 of 91) of the available FAs were not willing to participate. The impact of this selection bias is difficult to judge.

The impact of illicit drugs including anabolic steroids could be a contributing factor to the arrhythmias. The information of the exact intake and dosage of these drugs is hard to get. The percentage of FAs in our study admitting the use of amphetamines or other drugs was amazingly high. During the 1950s and 1960s, amphetamines were not illegal, yet. However, cardiotoxic drugs do not typically cause SND, but if anything tachyarrhythmias.[3]

Former FAs were still doing slightly more endurance training than the controls; however, the percentage of persons performing >4 h of cardiovascular training was similar.

This study is not aimed at comparing risks and benefits of performing high-endurance training at any level or comparing different levels. We chose not a sedentary control group to make the difference not too striking but a group of individuals performing a moderate amount of physical activity.[33]

Currently, we cannot make any recommendations how to identify endurance athletes who will develop SND in the long-term follow-up. Perhaps, in the future, we will recognize that in endurance athletes with a resting heart rate of <40 b.p.m. at day time, the likelihood of later SND is high and that these athletes might be encouraged to reduce or change their training program.

Conclusions

It is doubtful that long-term endurance level at a very high level induces only physiological and fully reversible changes of the

heart. The elderly athlete may not be as healthy as believed: among FAs, SND occurred significantly more often compared with age-matched controls. Also there is a trend towards more frequent atrial fibrillation/atrial flutter and ventricular tachycardias in FAs. Further studies have to evaluate the impact on how to advise the public on clinical follow-up and management of arrhythmias in former competitive endurance athletes.

Conflict of interest. none declared.

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CLINICAL VIGNETTE

doi:10.1093/eurheartj/ehm344
Online publish-ahead-of-print 7 August 2007

An unusual cause of dyspnoea in an 83-year-old woman

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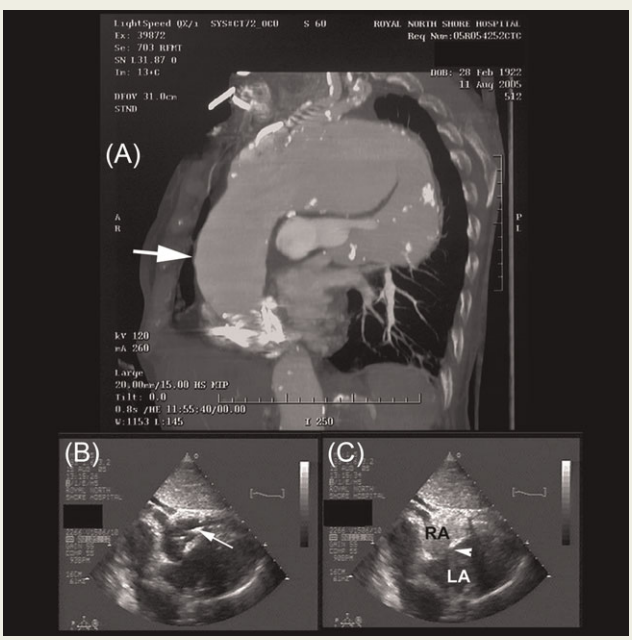
An 83-year-old woman was referred for assessment of rapidly worsening dyspnoea, limiting mobility to 10 yards. She had a history of multiple potential sources of her dyspnoea: coronary artery disease (previous stenting of the right coronary artery, a chronic total occlusion of the mid left anterior descending artery, and 50% stenosis in the circumflex with normal left ventricular function), chronic obstructive airways disease requiring inhaled bronchodilators and steroids, and chronic renal impairment with a mild anaemia. She had also been noted to have a stable aneurysm of the ascending aorta (4.5 cm in diameter) at last angiography 2 years before.

Examination at 45° revealed central cyanosis with oxygen saturation of 88% on room air but normal jugular venous pressure and clear lung fields to auscultation. On lying flat, the oxygen saturation improved to 92%. Ventilation perfusion lung scan indicated a low probability for pulmonary emboli. Chest X-ray showed widening of the superior mediastinum with the enlargement of the cardiac silhouette. Computed tomography (CT) aortogram showed an ascending aortic aneurysm with a maximum diameter of 6.8 cm (Panel A), substantially larger than the last measurement 2 years before. Transthoracic echocardiography showed a markedly dilated ascending aorta impinging on the right atrial free wall resulting in a functional tricuspid stenosis (mean gradient 5 mmHg), with contrast bubble study demonstrating a right-to-left shunt through a patent foramen ovale (PFO) (Panels B and C). Hyperoxic pulmonary function testing showed a shunt of 24%. The patient declined aortic surgery and was discharged on home oxygen, but agreed to consider palliative percutaneous PFO closure. However, 2 days later, she presented with chest pain. Repeat CT now showed dissection of the enlarging ascending aortic root aneurysm. The patient requested active measures be withdrawn and she died a few hours later.



This case illustrates an unusual mode of presentation for an ascending aortic aneurysm: marked hypoxia with cyanosis due to right-to-left intracardiac shunting resulting from right atrial compression. There were multiple other possible causes of dyspnoea including coronary ischaemia, left ventricular systolic or diastolic dysfunction, worsening of airflow limitation, anaemia, or fluid overload from chronic renal impairment. However, hypoxia with clear lung fields on examination and on chest X-ray suggested intracardiac right-to-left shunting as the cause.

Intracardiac right-to-left shunting can be interventricular but usually occurs across the atrial septum through an atrial septal defect or more commonly through a PFO and only when the right atrial pressure exceeds the left atrial pressure. Isolated elevation of the right atrial pressure is usually due to pulmonary hypertension but may also occur with obstruction to right atrial outflow (due to right atrial myxoma, tricuspid stenosis, or localized right ventricular dysfunction) and extrinsic compression of the right atrium (due to tumour, localized pleural or pericardial effusion, or, as in this case, due to aortic root enlargement).

Platypnoea-orthodeoxia syndrome (where dyspnoea and cyanosis are characteristically worse in the upright posture and improved by lying supine) is a rare condition usually where there is distortion of the interatrial septum, opening a PFO, which is exacerbated by erect posture. Distortion of the interatrial septum may result from right pneumonectomy/lobectomy or abnormal aortic root anatomy. Enlargement of the ascending aorta rotates the heart anti-clockwise and may result in anterior displacement of the superior limbus of the foramen ovale into the right atrial cavity, causing a PFO to be held open while compression of the right atrium raises pressure, promoting right-to-left shunting. The effects of gravity in the erect position cause further anterior and inferior displacement of the aortic root increasing PFO aperture size and right atrial compression resulting in increased shunting and worsening of hypoxia.

Panel A Computed tomography scan of the thorax in the sagittal plane demonstrating the ascending aortic aneurysm (arrow) with a maximum diameter of 6.8 cm.

Panel B Transthoracic echocardiogram from the subcostal view before bubble contrast injection showing the atrial pacing lead (arrow) in the right atrium, but no contrast in either atrium.

Panel C Similar view on transthoracic echo after bubble contrast injection showing right-to-left shunting across a patent foramen ovale (white arrow), as a result of the right atrial compression, with contrast filling the right atrium (RA) but also seen in the left atrium (LA).